The Impact of Aggregate and Idiosyncratic Income Shocks on Health Outcomes: Evidence from the PSID

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Abstract

In this paper, we investigate the impact of aggregate and idiosyncratic economic shocks on health using data on self-reported health status and mortality from the Panel Study of Income Dynamics. First, we document a large correlation between poor macroeconomic conditions and mortality for working-aged men. This correlation is robust to controls for baseline health which mitigates concerns that the correlation is the result of selection. There is no relationship between macroeconomic conditions and mortality for women. Next, to better understand how much of this correlation is the result of a causal impact of income shocks on health, we use methods from the literature on dynamic panel data models. Doing this, we find evidence of a causal impact of income shocks on health for working-aged men at the lowest parts of the income distribution. Finally, our analysis provides no evidence that recessions are good for your health.

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1 Introduction

The relationship between economic circumstances and health or the gradient has been the subject of academic inquiry for quite some time. While these inquiries have documented a strong positive correlation between socioeconomic status (SES) and health in a variety of contexts, they have failed to produce a consensus among scholars concerning the underlying causal pathways. Indeed, fierce debate has characterized the discussions among social scientists concerning the possible directions of causality with the dividing lines often being drawn between disciplines. Typically, on one side of the divide are the economists, who tend to champion the causal pathway from health to income (Smith 1999, Adams, Hurd, *et al.* 2002). On the other side of the divide are the public health experts and epidemiologists who tend to be advocates of the reverse causal pathway from SES to health (Marmot, *et al.* 1991, Marmot 2004). In this paper, we attempt to shed a new light on this debate by tackling the question of what happens to a person's health when their economic circumstances deteriorate.

To do this, we use data from the Panel Study of Income Dynamics (PSID) which offers a wealth of information which can be exploited to investigate this issue. To measure economic circumstance, we use data on income, labor supply and county-level unemployment rates. Our health data are provided by measures of self-reported health status (SRHS) and the PSID's death

file which provides a record of the deaths of all PSID respondents through 2003.

One primary advantage of the PSID is that its longitudinal structure allows us to use a rich literature on the estimation of dynamic panel data models. The estimation technique that we use from this literature comes from Arellano and Bond (1991). It exploits moment conditions which allow health to impact labor supply in contemporaneous and future time periods. If valid, these conditions enable us to identify the causal impact of income shocks on health. One of the advantages of the PSID is that its length guarantees a rich set of moments which allow us to carry out specification tests that shed light on the validity of these restrictions which are imposed on the data. In addition, the procedure allows for individual-specific fixed-effects which can be arbitrarily correlated with the right-hand-side covariates which mitigates many concerns of omitted variables bias. While this and similar techniques have commonly been employed in labor economics (see Carrasco 2001, Hyslop 1999, Meghir and Pistaferri 2004, for just a few examples), these techniques are utilized with far less frequency in health economics.

The first main finding of this paper is that we document a large correlation between poor macroeconomic conditions and high mortality among working-aged men. This finding is robust to a set of controls for baseline morbidity which, at least partially, mitigates concerns that the finding is the consequence of selection or economically depressed areas tending to be composed of unhealthy people rather than the direct causal impact of recessions on health. While there is a similar correlation among older men, it is substantially less pronounced. In addition, aggregate economic conditions in a given survey year tend to be highly correlated with mortality within one year, but not within three or five years of the survey year. Thus, if recessions have any causal impact on mortality, these effects occur rather quickly. Finally, we find no relationship

between recessions and mortality for women.

Our next main finding is that we provide some evidence of a causal impact of income shocks on the morbidity of working-aged men using the Arellano Bond estimator. Our estimated coefficient on income is large and is equal and opposite the coefficient on age. This suggests that part of the correlation between recessions and poor health for working-aged men is the consequence of an underlying causal relationship. These effects tend to be concentrated in the bottom part of the income distribution. However, it is important to mention that it is not clear how much of the estimated impact of an income shock on morbidity actually translates into an impact on mortality. Finally, once again, we do not find any relationship between income and health for women.

In addition to shedding some light on the gradient, these results also contribute to a vast and related literature on the association between and recessions and health. Initially, the conventional wisdom on this topic was that health deteriorated during economic downturns. Indeed, influential studies by Brenner (1971, 1975 and 1979) have shown that infant mortality and adult mortality from cardiovascular disease, cirrhosis, suicide and homicide exhibit strong counter-cyclical variation. However, these studies have been criticized by many (*e.g.* Kasl 1979; Wagstaff 1985; Cook and Zarkin 1986), particularly, on the grounds that they are tainted by omitted variables bias. Many studies (e.g. Forbes and McGregor 1984; McAvinchey 1986; Joyce and Mocan 1993) that address these criticisms cannot replicate Brenner's findings.

More recently, Ruhm (2000) has used panel data from US states and fixed-effects estimation to address this omitted variables bias.[1] Doing this, he shows that mortality rates from cardio-

[1]Similar findings have been uncovered in Spanish provinces (Tapia-Granados 2002) and in OECD Countries (Gerdtham and Ruhm 2002).

vascular disease, influenza/pneumonia and liver ailments are actually *pro*cyclical. Interestingly, Ruhm (2000 and 2005) also provides evidence that many risk factors for mortality from these causes also decrease in recessions. Using micro-data from the Behavioral Risk Factor Survey, he shows that diets become less healthy and that drinking, smoking and physical inactivity all increase when the economy improves.

However, there is a potential problem with Ruhm's results due to the fact that mortality rates in a state are typically measured with error which almost certainly will be correlated with the macroeconomic conditions which prevail in that state. The reason for this is that recessions tend to be accompanied by large out-migrations of people (Blanchard and Katz 1992). In the presence of migration out of a state, mortality rates are very difficult to compute because out-migration will decrease the number of deaths that occur in that state for purely mechanical reasons.[2] Although this increase in deaths may be small in absolute terms, it is apt to be large in percentage terms due to the infrequency with which death occurs. The net result is then a spurious correlation between poor macroeconomic conditions and low mortality. Our failure to uncover any evidence that health improves during an economic downturn using individual level panel data (which is unaffected by these issues) suggests that Ruhm's results may be driven by the impact of migration on the measurement of the mortality rate as opposed to the impact of recessions on the true mortality rate.

The balance of this paper is organized as follows. Section 2 discusses some theoretical considerations. In Section 3, we discuss the data. In Section 4, we investigate the relationship between aggregate economic shocks and health. In Section 5, we discuss the relationship between

[2] For more on this issue, see Abraído-Lanza, *et al* (1999) and Palloni and Arias (2003).

idiosyncratic economic shocks and health. Section 6 concludes.

2 Theoretical Considerations

In this section, we discuss the avenues through which recessions may impact a person's health within the context of a simple model of health investment *a la* Grossman (1972).[3] We start out by assuming that a person's health is given by some stock variable which we call H_t. Individuals have agency over their health in the sense that they can improve their health by investing i_t units of their time in its production.[4] We let w_t denote the time t wage and let $\theta(w_t)i_t$ be the health production function. We allow the parameter $\theta(w_t)$, which determines the marginal productivity of health investment, to depend on wages. We assume that $\theta' \geq 0$ so that higher wages either improve health or at the minimum have no impact on health. Finally, we assume that health evolves according to

$$H_{t+1} = \theta(w_t)i_t + (1 - \delta_t)H_t. \tag{1}$$

where δ_t the rate of depreciation.

Consumers maximize an inter-temporal utility function which depends on their health and their consumption of a commodity which we call c_t subject to their lifetime resource constraint. We let $u(c_t, H_t)$ be their sub-utility function and we assume that $u_c > 0, u_H > 0, u_{cc} < 0$ and

[3] The framework in this section is simple in that it abstracts from numerous factors which may influence health, many of which can be found in Grossman's original work and the literature which Grossman's work inspired. However, our aim in this section is not to add to the literature on the theory of health investment. Rather, it is to illustrate why one would expect economic shocks to have an ambiguous impact on health.

[4] One of the many contrasts between our simple formulation and Grossman's original formulation is that we only have one time input in the health production function.

$u_{HH} < 0$. If we let β denote the discount factor, the inter-temporal utility function is then given by

$$U = \sum_{t=0}^{T} \beta^t u(c_t, H_t) \tag{2}$$

where T is the length of life, which is endogenous in Grossman's framework, but exogenous in ours. We let l_t denote the consumer's labor supply at time t. In any time period, the consumer has finite time and, thus, the consumer's labor supply and health investment decisions are constrained by the relation $\iota_t + l_t = 1$. The consumer's lifetime resource constraint is then given by

$$\sum_{t=0}^{T} \frac{c_t}{(1+r)^t} + \sum_{t=0}^{T} \frac{w_t(1-l_t)}{(1+r)^t} = A_0 + \sum_{t=0}^{T} \frac{w_t}{(1+r)^t} \tag{3}$$

where A_0 is the consumer's initial asset level.

If we substitute equation (1) into equation (3), we can obtain

$$\begin{aligned} \sum_{t=0}^{T} \frac{c_t}{(1+r)^t} + \sum_{t=0}^{T} \frac{H_t}{(1+r)^t} \left[\frac{w_t}{\theta(w_t)}(r+\delta_t) \right] &= A_0 + \sum_{t=0}^{T} \frac{w_t}{(1+r)^t} \\ -\frac{w_T}{\theta(w_T)}(1+r) \left[\frac{H_{T+1}}{(1+r)^{T+1}} - H_0 \right]. \end{aligned} \tag{4}$$

An almost identical manipulation can be found in Case and Deaton (2004). This form of the lifetime budget constraint shows how the consumer's lifetime resources are spent on the elements of the utility function: consumption and health. An interesting feature of equation (4) is that it delivers the user cost of health which is $\frac{w_t}{\theta(w_t)}(r+\delta_t)$. As pointed out by Case and Deaton (2004), the user cost of health tells us the rate at which health can be converted into cash in

order to pay for consumption. Using this form of the budget constraint, it is trivial to show that the first order conditions for health and consumption together imply

$$\underbrace{\frac{u_H(c_t, H_t)}{u_c(c_t, H_t)}}_{MRS} = \underbrace{\frac{w_t(r + \delta_t)}{\theta(w_t)}}_{\psi_t}. \tag{5}$$

This condition is an intra-temporal relation which governs the optimal division of time between work and health investment by equating the consumers willingness to substitute consumption for health with ψ_t or the user cost of health.[5]

The impact of a decline in the wage on health will largely depend on its impact on the user cost of health. This impact can be decomposed into two separate effects as follows

$$\frac{\partial \log \psi_t}{\partial \log w_t} = \frac{1}{w_t} - \frac{\theta'(w_t)}{\theta(w_t)}. \tag{6}$$

The first term on the right-hand side is positive so that higher wages will discourage health investment by increasing its time cost. The second term tells us that, provided that $\theta' > 0$ (which we concede is a controversial assumption in the economics profession), higher wages will increase the demand for health by improving the marginal productivity of health investment.[6,7]

[5] The other optimality condition is $u_c(c_t, H_t) = (1 + r)\beta u_c(c_{t+1}, H_{t+1})$ which is the standard inter-temporal Euler equation in which the marginal utility of consumption today is equated to the discounted marginal utility of consumption tomorrow.

[6] The interpretation of $\delta_t + r$ is straight foward in equation (5). A high rate of depreciation implies that the net returns to health investment are low which discourages health investment. Higher interest rates imply that consumption tomorrow relative to today is cheap. This creates incentives to work more today and save for tomorrow which entails less health investment today.

[7] Had we also considered the possibility of endogenous death, higher wages would also increase the returns to a longer life and, thus, create an additional incentive for the consumer to invest in their health. Thus, with endogenous mortality, we would have a third channel through which wages would impact the demand for health. The relative magnitude of this effect would depend crucially on the consumers discount factor with the effect being less important for more myopic consumers.

Clearly, the net impact of a decline in the wage will depend on the relative magnitudes of both of these effects.[8] The question of which effect dominates is an empirical matter and the subject of the remainder of the paper.

3 Data

The data that we employ come from the PSID. Our sample includes variables on age, race, education, self-reported health status (SRHS), the unemployment rate in the respondent's county of residence, labor income and mortality. Table 1 reports the summary statistics for all of the variables in our sample except for the mortality data. Note that because we include the Survey of Economic Opportunities in our sample, which we discuss in more detail later, these summary statistics may not be representative of the US population.

All of our data other than labor income span the years 1984 to 1993. The reason for this is that the SRHS data are not available prior to 1984, whereas the unemployment data are not publicly available past 1993. We employ labor income data from 1978 to 1993. The reason

[8] It is informative to contrast the impact of wages on health in our framework with what happens in Grossman's original model. In this setup, we allow the health production function to depend on the wage which suggests the possibility of a direct causal effect of wages on health. This is a departure from much of the health economics literature. The reason is that while many non-economists have come to accept that income or social status may directly cause health (see Marmot, *et al.* 1991, Marmot 2004 for example), economists have tended to be less accepting of direct causation from income to health. (see Smith 1999 and Adams, Hurd, *et al.* 2003, for example). In contrast, in Grossman's original setup, in which medical care and time are inputs in the health production function and in which sickness reduces a person's ability to work, an increase in wages will raise the value of productive time which will then create an incentive for people to further invest in their own health by substituting away from the time input towards medical care. Consequently, while it is the case in Grossman's framework that richer people are also healthier people, it is only because higher wages increase incentives for health investment. In contrast, in this setup, if higher wages lead to better health, it is only because we allow the wage to directly impact the marginal productivity of health investment. Practically speaking, this distinction is not important for our purposes. What is important is that there are pathways through which an increase in the wage will either improve or deteriorate one's health. Our choice of modeling the positive impact of wages on health by allowing the wage to directly enter the health production function was only to simplify the model.

for going back to 1978 with these data is that it allows us to have more instruments when we employ a GMM procedure from Arellano and Bond (1991) later on in the paper. The addition of more instruments enhances the efficiency of the procedure. Additional detail concerning this is provided in Section 5.

The PSID contains a sample of economically disadvantaged people called the Survey of Economic Opportunities (SEO). There is little consensus within the profession about how one should deal with the SEO. Because it is selected on income and, thus, endogenous, conventional weighting schemes will not work. Accordingly, some people such as Lillard and Willis (1977) simply recommend dropping the SEO due to endogenous selection. Nevertheless, there are others such as Hyslop (1999) and Meghir and Pistaferri (2004) who include the SEO. We follow these authors and include the SEO as well. Our reason for its inclusion is that one of our primary outcomes is mortality which happens relatively infrequently, especially for younger people. Consequently, dropping the SEO from the sample would have resulted in too few deaths in our data.

Our mortality data come from the PSID's death file which is considered sensitive data and, thus, not publicly available. The death file contains mortality information on all individuals in the PSID from 1968 to 2003 who were known to have died prior to 2003. There are a total of 4705 recorded deaths in the file. Because it is essential for our purposes to control for the individual's morbidity and because SRHS is not available prior to 1984, we only use death dates from 1984 to 2003. This reduces the number of deaths to 2849. Mortality information first comes from interviews with PSID families. PSID then corroborates this information with the National Death Index. For some individuals, PSID could not precisely date the time of death. We do not use these less precise dates. This further reduces the number of deaths in our data

to 2797.[9]

We assume that all individuals who were not in the death file were alive as of 2003. We concede that this is a strong assumption. However, there was an effort on the part of PSID to verify the deaths of people in families who have attrited from the panel. Nevertheless, there may still be some people who have left the panel and, subsequently, died after the household was being interviewed by PSID and, thus, are not in the death file. This would create a situation in which dead people are mis-classified as alive. To address this issue, in addition to working with the full sample, we also work with a restricted sample of people that includes only people who were known to have died prior to 2003 and people who were present in the 2003 wave of the PSID and, thus, known to have survived to that year. Our core results are unaffected by restricting the sample in such a way. Because of this, we decided to work with the full sample for most of our results as this gave us more information and, therefore, higher efficiency.

Table 2 summarizes our mortality data. The first column of the table reports the percentage of people 30 years or older that have died before 2003 for each wave of our sample. For example, the table shows that 22.83% of all people who were older than 30 in 1984 have died prior to 2003. The second column shows the average number of years that each individual has survived subsequent to the survey year. Because PSID records both the year and month of death, survival is measured to the nearest month. If the person had not died by 2003, we censor the observation at 2004 minus the survey year. For example, the people from the 1984 wave who survived until 2003 have their observations censored at 20 years.

[9] It is important to highlight that while this may appear to be a large number of deaths, it actually can become quite small once we start to look at sub-samples of the PSID which are broken down by gender and age. For example, in the 1984 wave, there were 315 recorded deaths of men who were between the ages of 30 and 60 at the time of the survey. However, if we were to have excluded the SEO, this number drops to 132 which highlights the importance of including the SEO in our analysis.

Figure 1 plots survivor functions from the PSID for men between the ages of 30 and 60, men older than 60, women between ages 30 and 60 and women older than 60. Each panel of the figure contains ten graphs which correspond to each of the ten years of our panel (*i.e.* 1984 to 1993). Each of these graphs takes all of the people in the sample of a certain age from a given wave of the survey and plots the percentage of these people who survived to each subsequent year through 2003. For example, the bottom graph in each panel corresponds to the base year 1984 and plots the percentage of people who survived until 1985, 1986, 1987, *etc.*[10]

The figures show that, while in a particular survey year death is a low probability event, over the course of 20 years, it is quite common. For men and women, we see that 10% of the baseline sample who was younger than 60 in 1984 died prior to 2003. For people who were older than 60 in 1984, the probabilities become 70% and 60% for men and women, respectively.

Our other measure of health is SRHS which is a categorical variable that takes on integer values between one and five and measures the respondent's assessment of their own health. A one represents excellent health and a five represents poor health. If SRHS is two, three or four then the respondents rate their health as very good, good or fair. These measures, while subjective, do correlate extremely well with more objective measures of health. Numerous studies have shown that SRHS is informative of specific morbidities and subsequent mortality (Mossey and Shapiro 1982; Kaplan and Camacho 1983; Idler and Kasl 1995). In addition, Smith (2004) has used retrospective health measures from the PSID and shown that there is a tendency for people to downgrade their self-assessment of their own health when a new condition manifests.[11]

[10] It is important to note that our data show the stylized fact that women have lower mortality, as shown in Figure 1, and higher morbidity, as shown in Table 1. However, this does *not* suggest that the SRHS are of poor quality. Rather, it merely reflects that women tend to suffer from a different distribution of chronic ailments than men (Case and Paxson, 2005).

[11] Since the retrospective health measures are only available after 1999 and because our sample only goes through

Throughout this analysis, we map the SRHS measure into two dummy variables: good health, which is turned on when SRHS is either a one or a two, and bad health, which is turned on when SRHS is either a four or a five. The omitted SRHS category is good health which is coded as SRHS being equal to three.

Table 3 shows the results from estimation of Cox-Proportional hazard models to illustrate the relationship between SRHS and mortality in the PSID.[12] We estimate the models using the 1984 wave of the PSID with the number of years that the individual survived subsequent to 1984 as the dependent variable. If the individual did not die prior to 2004, then the outcome is censored at 20. As can be seen in the upper and bottom left panels, good health is associated with a lower probability of death, whereas bad health is associated with a higher probability of death for men and women between the ages of 30 and 60. It is important to note that the whole range of the SRHS measure appears to be informative of mortality, although the bottom two categories (*i.e.* bad health) seem to be the most informative. Turning to men and women older than 60 in the right-hand panel, we see that SRHS remains important. However, now we see that only good health is informative of male mortality, whereas bad health is only informative of female mortality for this sample of older people. This table provides further evidence that the SRHS variables in the PSID are very good measures of the respondent's health.

A final issue with our data that warrants some attention concerns the possibility of correlated disturbances within counties. As has been pointed out by many, such as Moulton (1990), in geographically stratified samples, estimation of standard errors can be problematic if there

1993, the retrospective health measures would not be well-suited for our purposes due to problems associated with recall bias.

[12] For additional detail on this estimation procedure, see Section 4.1.

are correlated disturbances within strata. Often times this critique is raised when aggregate variables (like county-level unemployment rates) are combined with micro-units. Failure to address this will tend to result in underestimated standard errors. However, because the PSID is not geographically stratified by county, it is not clear that Moulton's critique is relevant in our case. Indeed, if the sample is independent within counties, there is no need to adjust the standard errors at all. On the other hand, if there are correlated disturbances within counties, it would be necessary to adjust the standard errors regardless of whether or not county-level variables are being used. In other words, using a county-level variable with an individual unit does not change the survey design. Nevertheless, there still may be some concern that there are unobserved correlated factors within geographic units which affect our standard errors. A solution to this problem which is commonly employed in the profession is to cluster the standard error by county, although the asymptotic justification for such a procedure is not that clear in non-linear models. Unfortunately, county of residence is considered sensitive by PSID and, thus, is not publicly available. However, when using a single wave of the PSID, it is possible to construct a variable that is unique for all counties in a given state with the same unemployment rate. For example, if a person lives in a state which is coded as 12 and the unemployment rate in their county is 6, then we code this variable as 1206. It is important to state that this procedure is apt to be too conservative since it clusters on all counties in a given state with the same unemployment rate. Because unemployment rates change across time, we are unable to use this procedure when multiple waves of the sample are used. In practice, what we find is that our conclusions are unaffected by whether or not we use the clustering procedure.

4 Aggregate Economic Conditions and Health

We now turn to the task of investigating the relationship between aggregate economic shocks and health outcomes. In Section 4.1, we present cross-sectional evidence. In Section 4.2, we discuss the impact of mis-classified mortality on our cross-sectional estimates. In Section 4.3, we present panel evidence.

4.1 Cross-Sectional Estimates

We begin this section by estimating hazard models to investigate the relationship between unemployment and mortality in the 1984 wave of the PSID. We use the 1984 wave because using the earliest wave of the PSID that is available provides the highest variation in subsequent survival. In Section 4.3, we conduct an analysis that combines all ten waves.[13] We let T denote the number of years (measured to the nearest month) that an individual has survived subsequent to 1984. Because we only have mortality information through 2003, T is censored at 20 years.

We specify a proportional hazard function of the form

$$\lambda_i(t|x_i) = \exp(x_i\beta)\lambda(t) \tag{7}$$

where $\lambda(t)$ denotes the baseline hazard. x_i contains age, race, education, bad health (*i.e.* SRHS is either fair or poor), good health (*i.e.* SRHS is either excellent or good), state dummies and

[13] Estimating a hazard model with all ten waves of the panel would require the use of duration models with time varying regressors. These techniques raise many subtle issues which according to Lancaster (1990) have not been "fully clarified" in the literature. Consequently, we estimate duration models on single waves of the panel and reserve the full use of the panel for later when we use more transparent techniques that will yield results that are easier to interpret.

the county unemployment rate. The hazard function gives us the instantaneous probability of dying conditional on having survived t years.[14] We use the Cox Partial Likelihood Estimator to estimate β. This procedure does not require a specification for the baseline hazard. For more information on this estimation procedure, we refer the reader to Chapter 11 of Amemiya (1985). In practice, we experimented with other parametric hazard functions such as a Weibull, Exponential and Log-Normal specifications. Our conclusions were unaffected.

At this point in the paper, we do not concern ourselves with addressing unobserved heterogeneity. Typically, in duration models, addressing unobserved heterogeneity is more of an issue when attempting to identify duration dependence. As pointed out by Wooldridge (2002), the case for the explicit modeling of unobserved heterogeneity is "less compelling" when the researcher is interested in estimating the impact of a covariate of interest on mean duration. Nevertheless, there still may be some concern that there are unobserved individual characteristics which are correlated with mortality but differ systematically across areas with high and low unemployment. For example, if it is the case that healthier people are better able to move out of depressed areas to find work than unhealthy people, one would expect to see a correlation between high unemployment rates and poorer health outcomes. This mechanism suggests that any positive correlation between unemployment and bad health would at least partly reflect selection. However, it is our opinion that it is inappropriate to address this correlation with single spell duration data since identification would hinge upon a parametric assumption on the relationship between unobservables and the exogenous covariates.[15] Consequently, we address these

[14] Formally, we have that

$$\lambda_i(t|x_i) = \lim_{h \downarrow 0} \frac{P(t \leq T < t+h | T \geq t, x_{i,t})}{h}.$$

[15] Existing non-parametric methods for dealing with unobserved heterogeneity in duration models such as Heck-

selection concerns later on in the paper when we employ some fixed-effects estimation procedures in Section 4.3 and Section 5. These procedures have the advantage that they impose less stringent assumptions on the unobserved heterogeneity which, in turn, results in more transparent identification.

In Table 4, we report estimates of hazard models using a sample of men and women between the ages of 30 and 60. Each cell reports the hazard ratio associated with raising a given covariate by one unit and the t-statistic corresponding to the underlying coefficient. If the hazard ratio is above unity, then the variable has a positive effect on mortality. It it is less than unity, then it has a negative effect. For men, we see that higher unemployment is associated with higher mortality in all five columns. Typically, the effect of unemployment on mortality is at least 50% the effect of age. However, when we look at working-aged women in the bottom panel, we see that the relationship between unemployment and mortality is flat in the first four columns and is moderately significant and negative in column 5.

The positive relationship between unemployment and mortality that we uncover for working-aged men most likely reflects two components: a selection component and a causal component. The selection component could result from unhealthy people tending to live in depressed areas, whereas the causal component may result from shocks to the economy adversely impacting people's health. However, the fact that positive effect of the unemployment rate is robust to the inclusion of controls for baseline health status, at least, partially mitigates the concern that these results are entirely being driven by the selection component.

In Table 5, we estimate probits using the same sample that was used to generate the results in

man and Singer (1984) typically assume independence between the unobserved heterogeneity and the exogenous covariates.

Table 4 where the dependent variable is an indicator for having died at any point between 1984 and 2003. The table reports the marginal effects. The results reinforce the results from the hazard models. We see that an increase of 1% in unemployment raises the probability of dying within twenty years by 0.004 points. It is interesting to contrast this with Table II from Ruhm (2000) where he showed that a 1% increase in the unemployment rate *lowers* the probability of dying *in that year* by *at least* 0.004 points. Thus, our estimated correlation between recessions and health for working-aged men is of a much smaller magnitude and of the opposite sign than Ruhm's estimates.

Table 6 estimates hazard models for men and women 60 years of older. Now we see a smaller effect of recessions on health for men. The hazard ratios are a little more than half of what they were in Table 4. Once again, we do not see much evidence of any relationship between recessions and health for women.

4.2 Mis-Classified Mortality

Table 7 reports estimates of the Cox-Proportional Hazard model for a restricted sample of the data which only includes people whose deaths were recorded in the death file or people who were present in the panel in 2003 and, thus, known with absolute certainty to have survived to that year. We estimate the models for working-aged men and women. Accordingly, Table 7 is comparable to Table 4. Restricting the sample in this way excludes any people who are actually dead but not present in the death file from the sample. In many respects, this procedure is akin to chemotherapy in the sense that, while it does purge the data of mortality data that is mis-classified (*i.e.* dead people who are coded as alive in our sample), it also purges the data of

far more mortality data that is correctly classified. In this sense, this is somewhat of a draconian solution to the problem of mis-classification.

Comparing the results in Tables 7 and 4, we see few differences. In fact, for men, the hazard ratios for the unemployment rate are virtually identical in both tables. The only difference is that the standard errors on the unemployment estimates are higher in Table 7, which is not surprising given that the sample in that table is 40% smaller. Turning to women, we see that the hazard ratios for the unemployment rate are all systematically less than unity and are all lower than they are in Table 4. However, unemployment is only significant in column 5. Overall, the similarities in Tables 4 and 7 suggest that possible mis-classified mortality data is not affecting our estimates in any meaningful way.

In Table 8, we estimate probits using a sample of working age men and women from the 1984 wave where the dependent variable is a selection indicator which was turned on if the person was present in the restricted sample in Table 7. What we see is that, for both men and women, in all five specifications that we considered, unemployment is never significant. This suggests that there is no systematic relationship between observations for which mortality may be mis-classified and the unemployment rate. This further mitigates any concerns that the results in Tables 4, 5 and 6 may be adversely impacted by a failure of PSID to document the deaths of every person who attrites.

4.3 Panel Estimates

We begin this section by considering models of the form:

$$d_{i,t}^{j} = 1\left(x_{i,t}\eta_{R}^{j} + \alpha_{i}^{j} + \varepsilon_{i,t}^{j} \geq 0\right) \text{ for } j = 1, 2 \text{ and } 3 \tag{8}$$

where $d_{i,t}^{1}$, $d_{i,t}^{2}$ and $d_{i,t}^{3}$ are indicators for dying within one, three and five years of the survey year. We estimate the parameters of the model using a random effects probit estimator. These models have the advantage that they make efficient use of all ten waves of our PSID sample and avoid the complications of duration models with time-varying regressors.

Tables 9 and 10 report the random effects estimates of equation 8 for men and women respectively. The top panels corresponds to people between the ages of 30 and 60 and the bottom panels corresponds to people who are older than 60. In Table 9, two points are worth noting. First, the unemployment rate is only a positive and significant correlate of mortality when the outcome is dying within one year of the survey year. This is true of both working-aged and older men. Second, we see that the point-estimate of the unemployment coefficient in column 1 is 0.038 for working-aged men and 0.029 for older men so that the relationship between unemployment and mortality is much stronger for working-aged men than for older men. Turning to Table 10, we see no significant relationship between unemployment and mortality for women. Thus, both tables reinforce the conclusion of Section 4.1 that a positive relationship between recessions and mortality is only present for men and is strongest in the population of working-aged men. In column 3 of the table, there is some weak evidence of a negative relationship between recessions and mortality for women, but the coefficient is only moderately significant with a t-statistc of -1.41.

Next, we consider models of the form:

$$h_{i,t}^{j} = 1(x_{i,t}\eta_{F}^{j} + \alpha_{i}^{j} + \varepsilon_{i,t}^{j} \geq 0) \text{ for } j = B, G \tag{9}$$

where $h_{i,t}^{B}$ is an indicator for bad health (SRHS greater than or equal to four) and $h_{i,t}^{G}$ is an indicator for good health (SRHS less than or equal to two). We estimate the parameters in equation 9 using the conditional logit model. This procedure maximizes the likelihood function conditional on the sum of $h_{i,t}^{j}$ which is a sufficient statistic for α_{i}^{j} when $\varepsilon_{i,t}^{j}$ has a logistic distribution and the covariates are strictly exogenous. Consequently, this conditioning procedure purges the model of the fixed-effect, α_{i}^{j}. Its advantage is that it does not require any distributional assumptions on the fixed-effect and, thus, allows for arbitrary correlation between α_{i}^{j} and $x_{i,t}$.[16] This offers a semi-parametric way of addressing concerns that the previous results in this paper were the consequence of selection resulting from depressed areas being inhabited by less healthy people rather than the direct causal effect of recessions on health. Of course, as is always the case, the benefits of relaxing distributional assumptions come at the expense of reduced efficiency.

The conditional logit results are presented in Table 11. We present estimates for four groups: working-aged men and women and older men and women. Because the conditioning procedure purges the model of all time invariant variables, the need for a rich set of covariates is obviated. Accordingly, we only include age and the unemployment rate as control variables. When bad health is the dependent variable, there is no relationship between recessions and health. Presumably, however, this has a lot to do with the infrequency with which the bottom

[16] For a textbook treatment of the conditional logit model, we refer the reader to pp. 839 - 840 of Greene (2000).

two SRHS categories occur in the sub-sample of working-aged people. Indeed, this can be seen in the "effective" sample sizes in the table.[17] When good health is the outcome, the effective sample sizes are 2826 and 3076 for men and women, respectively, and when bad health is the outcome the effective sample sizes drop to 1307 and 1714, respectively. However, for working-aged men, when good health is the dependent variable, we see that increases in the unemployment rate are associated decreases in health status, but for the remaining three sub-groups in the table, there is no relationship between recessions and health.

5 Idiosyncratic Economic Conditions and Health

We begin this section by considering the relationship between macroeconomic conditions and an individual's economic circumstances. To illustrate this relationship, we estimate linear fixed-effects regressions where the dependent variable is either (log) labor income or an indicator for having positive labor income which we sometimes call labor supply for lack of a better name. The independent variables are the unemployment rate and a quadratic in age. These regressions give us some notion of how variation in macroeconomic conditions across time for a given individual translates into an individual-specific earnings shock. The results are reported in Table 12. In the table, we do not see any substantial effects of unemployment fluctuations on labor supply, but we do see large effects on labor income. The estimates indicate that a 1 point increase in the unemployment rate translates into a reduction of earnings of approximately 3% on average. We now focus our attention to the task of identifying the impact of these individual-specific earnings

[17] The condition logit model only uses observations where the dependent variable switches states. In our case, this means people have at least one transition into or out of a health state. The "effective" sample size refers to the number of people in the sample who have at least one transition into or out of a state.

shocks on health outcomes.

To do this, we work with the dynamic model:

$$h_{i,t}^{B} = \alpha_i + \gamma h_{i,t-1}^{B} + y_{i,t}'\lambda + a_{i,t}\delta + \upsilon_{i,t}. \tag{10}$$

$h_{i,t}^{B}$ is an indicator for bad health (*i.e.* SRHS is either fair or poor). $y_{i,t}$ is a vector which includes income and labor supply (an indicator for having zero labor income). $a_{i,t}$ is age. We assume that the residual is mean zero and serially uncorrelated so that $E[\upsilon_{i,t}] = E[\upsilon_{i,t}\upsilon_{i,s}] = 0$ for $s \neq t$.[18] To purge the model of fixed-effects, we work with a first-differenced version of (10):

$$\Delta h_{i,t}^{B} = \gamma \Delta h_{i,t-1}^{B} + \Delta y_{i,t}'\lambda + \Delta z_{i,t}'\delta + \Delta \upsilon_{i,t} \tag{11}$$

Equations (10) and (11) account for two important aspects of the theory of health investment. First, because equation (11) is purged of the fixed-effect, it allows for all time-invariant individual characteristics to be correlated with both health and earnings. This is important since heterogeneity in preferences, discount factors and budget constraints will generate a spurious correlation between earnings and health and, thus, it is essential that the model is purged of these unobserved individual characteristics. Second, because we control for an individual's health yesterday, we rule out any omitted variable biases that would result from a person's health yesterday feeding-back and impacting labor supply today. This is particularly important in light of Grossman's original formulation of health investment in which sickness reduces a person's stock of "healthy time" which, in turn, constrains their ability to earn. In fact, the estimation proce-

[18] We will provide tests of the plausibility of the lack of serial correlation in $\upsilon_{i,t}$ later in the paper.

dure that we employ, which is discussed in the next sub-section, can be generalized to allow for, not only health yesterday, but also health today, to impact today's earnings.

5.1 Identification and Estimation

Identification of the parameters in equations (10) and (11) comes from two sets of moment conditions which exploit the time dimension of the data. Adopting the notation that $x_i^t = (x'_{i,1}, ..., x'_{i,t})$, the strongest set of moment conditions that we employ is

$$E^*[\upsilon_{i,t}|h_i^{t-1}, y_i^t] = 0 \tag{P}$$

where $E^*[y|x]$ denotes the linear-projection of y onto x. We call this Assumption P because these moment conditions suppose that income and labor supply are predetermined variables. This condition assumes that health shocks today are uncorrelated with the history of health outcomes through yesterday and labor market outcomes through today. However, it allows for feedback in the sense that health today can impact labor market outcomes tomorrow. The weaker set of moment conditions that we work with is

$$E^*[\upsilon_{i,t}|h_i^{t-1}, y_i^{t-1}] = 0. \tag{E}$$

We call this Assumption E because, in contrast to Assumption P, it allows for a contemporaneous relationship between health and labor supply and, thus, treats income as an endogenous variable. Assumption E has the advantage that it imposes weaker assumptions on the data, but comes at

the expense of reduced efficiency.[19]

To estimate the model, we use the GMM estimator outlined in Arellano and Bond (1991). Arellano and Bond (AB) applies Assumptions P and E to the first-differenced model in equation (11) and, thus, uses

$$E^*[\Delta v_{i,t}|h_i^{t-2}, y_i^{t-1}] = 0 \tag{12}$$

and

$$E^*[\Delta v_{i,t}|h_i^{t-2}, y_i^{t-2}] = 0 \tag{13}$$

as moment conditions. Equation 12 applies Assumption P to the first-differenced model and, thus, uses y_i^{t-1} and h_i^{t-2} as instruments for $\Delta y_{i,t}$ and $\Delta h_{i,t-1}$. Analogously, equation 13, which is implied by Assumption E, uses y_i^{t-2} and h_i^{t-2} as instruments for $\Delta y_{i,t}$ and $\Delta h_{i,t-1}$. We follow the recommendations of AB and report the parameter estimates from the one-step procedure. As we discussed in the data section, the SRHS data is not available prior 1984 and, consequently, we can only use health as an instrument through that year. However, because data on labor income are available for the entire duration of the PSID, we employ data on income through 1978. We did not use data prior to 1978 because we did not expect income from 1977 or earlier to have much explanatory power for the first-difference in health from 1986 or later.

We investigated the possibility that these instruments are weak. Recent research has shown that when instrumental variables do not have sufficient explanatory power in the first-stage regressions, the finite sample distribution of the estimator can differ substantially from its asymptotic distribution (see Staiger and Stock (1994) and Bound, Jaeger and Baker (1995), for

[19] For an excellent discussion of using these types of moment restrictions to identify dynamic linear panel data models, see Arellano and Honoré (2001).

example). To look into this issue, we regressed $\Delta h_{i,t}$ and each element of $\Delta y_{i,t}$ on the vector, $(h_{i,t-2}, ..., h_{i,t-4}, y'_{i,t-2}, ..., y'_{i,t-4})$. The F-tests of joint significance of the regressors all had extremely low p-values and, thus, there was no indication that weak instruments was a problem. The results are not reported, but are available upon request.

5.2 Specification Tests

One of the primary advantages of the AB procedure is that the model's assumptions yield many moment restrictions which can be used to construct specification tests which shed light on the plausibility of the identifying assumptions of the model. AB propose two specification tests. The first test centers on the fact that when $\upsilon_{i,t}$ exhibits no serial correlation, we will have that $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}] \neq 0$ (provided that the process for $\upsilon_{i,t}$ does not have a unit root) and $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}] = 0$. This specification test calculates the sample analogues of $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}]$ and $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}]$ to construct statistics that converge to a standard normal distribution. We follow the notation in AB and let m_1 denote the statistic that is based on $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}]$ and let m_2 denote the statistic that is based on $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}]$.[20] Calculation of m_1 is very important because if $\upsilon_{i,t}$ follows a random walk then we will have that $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}] = E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}] = 0$. Consequently, it is possible for m_2 to be small even if $\upsilon_{i,t}$ exhibits a large degree of persistence. So, if the model is correctly specified and there is no serial correlation in $\upsilon_{i,t}$ then m_1 should be big and m_2 should be small. Further detail on the calculation of m_1 and m_2 can be found in AB (pp. 281 - 282).

The second specification test that we work with is the Sargan test of over-identifying restric-

[20] In fact, AB can accommodate serial correlation in $\upsilon_{i,t}$ of the form MA(q) via weaker moment conditions. However, as it turns out, our calculations of m_2 suggests that such accommodation is not necessary.

tions (Sargan 1958; Hansen 1982). We use the two-step Sargan Statistic which is robust to heteroskedasticity.[21] We chose the two-step statistic over the one-step statistic because Monte Carlo experiments in AB suggest that there is a tendency for the non-robust test to over-reject and, thus, AB recommend placing more weight on the two-step statistic. The statistic is asymptotically chi-squared with degrees of freedom equal to the number of over-identifying restrictions in the model.

5.3 Results

Tables 13 and 14 report the AB estimates for working-aged men and women, respectively. The top panel uses Assumption P and the bottom panel uses Assumption E. The first three columns use bad health as the dependent variable. The last column uses the five point categorical SRHS variable as the dependent variable. We concede that the linear model that we estimate does not allow for the ordinal nature of the five-point SRHS variable. However, the five-point SRHS variable has the advantage that it has more variation in the time-series than bad health which only changes when people move in or out of the bottom two SRHS categories. This increased variation in the time-series is extremely useful with fixed-effects estimation.

Table 13 provides evidence of a causal effect of income shocks on health outcomes for working-aged men. In the top panel, income and labor supply are significant in columns 1, 2, and 4. In the bottom panel, the only significant variable is labor supply in column 4. However, it is immensely important to emphasize that the point-estimates in columns 1, 2 and 4 in the bottom panel are always at least as large in magnitude as they are in the top panel. What this suggests is

[21] Unlike the Sargan Statistics, the specification test that uses m_1 and m_2 is defined in terms of any consistent estimator. In other words, the statistics m_1 and m_2 do not necessarily require the efficient two-step estimator.

that the low t-statistics in the bottom panel of the table are most likely the result of inefficiencies associated with the weaker set of moment conditions which come from Assumption E. Finally, in column 1 of both panels, we see that the coefficient on income is roughly equal and opposite the coefficient on age suggesting that the impact of an earnings shock on health is of a meaningful magnitude.

The specification tests in the bottom of both panels suggest that our moment restrictions hold up reasonably well in the data. Looking at the calculations of m_2, we cannot reject the null that $E[\Delta v_{i,t}\Delta v_{i,t-2}] = 0$ at the 5% level in all eight specifications. The p-values on m_1 are all extremely low and, thus, always reject the null that $E[\Delta v_{i,t}\Delta v_{i,t-1}] = 0$. This rules out a unit root in the process for $v_{i,t}$. The two-step Sargan Statistic, which AB recommend, is not significant at the 5% in columns 2 and 3 of both panels and is not significant at the 10% level in column 2 of both panels. It only has an extremely low p-value in the fourth column of both panels. However, this is not shocking since the linear model is probably not the best way to deal with the five-point SRHS variable.

Table 14, which reports the results for working-aged women, is a stark contrast to Table 13. Most of the specifications suggest that there is no relationship running from income shocks to health outcomes. However, in column 4 of the top panel, there is some weak evidence that an exogenous reduction in labor supply is associated with worse health. The estimates in column 3 of the bottom panel are the only estimates that are significant at conventional levels and are somewhat difficult to interpret. The coefficient on labor supply suggests that a negative shock to labor supply is bad for health, whereas the coefficient on income suggests that a negative income shock is *good* for health. The specification tests in the table perform reasonably well. Overall,

the table does not provide strong evidence of any effect of income shocks on health outcomes for working-aged women.

Finally, we investigate at which part of the income distribution the impact of income shocks on health is greatest. To do this, we construct dummies for being below the 25th and 75th percentiles of the income distribution. We estimate equation (11) under the assumption that these income variables are predetermined using the sub-sample of working-aged men and women. The quartiles that were used to construct the dummies were calculated separately for men and women.

The results are reported in Table 15. The specifications assume that income is predetermined. The first three columns correspond to men and the last two correspond to women. Columns 1 and 4 only include a dummy variable for having zero labor income and are, thus, identical to column 2 in the upper panel of Tables 13 and 14. Because approximately 25% of working-aged women in our data reported zero labor income, we do not include the 25th percentile dummy for women. In columns 1 and 4, we see that a shock to labor supply has a significant adverse impact on men's health and no impact on women's health. In columns 2 and 3, we include the 25th and 75th percentile dummies, respectively. We see that transitions into and out of these parts of the income distribution have no impact on men's health. The 75th percentile dummy is also insignificant for women. We conclude that, to the extent that this sections suggests that there is a causal impact of income shocks on men's health, the effects tend to be concentrated in the bottom part of the income distribution.

6 Conclusions and Caveats

In this paper, we documented a strong correlation between economic shocks and health for working-aged men. For this sub-population, we showed that county-specific macroeconomic conditions are strong predictors of mortality. This correlation persists even after controls for baseline morbidity are included in the regressions which partially mitigates concerns that the result is driven by the selection of less healthy people into depressed counties. In addition, we utilized a methodology from the literature on dynamic panel data models which allowed for the impact of health on labor supply and provided evidence that idiosyncratic shocks to income cause a deterioration in health status for those who resided in the lowest part of the income distribution. This provided an additional piece of evidence that part of the correlation between health and aggregate shocks that we observed for working-aged men is driven by an underlying causal relationship at least at the bottom part of the income distribution.

It is important to place these findings within the context of some of the literature which has investigated causal pathways between SES and health. One of the most important papers on this topic is Adams, Hurd, *at al.* (2003) who investigate causality between wealth and health in a population of older Americans. They find no evidence of a causal link from SES to mortality and many morbidities, but they do reject the hypothesis of non-causality for some primary causes of death of older men such as cancer and heart disease.[22] In a related piece, Meer, Miller and Rosen (2003) use inheritance as an instrument for changes in wealth and find no evidence that health improves with exogenous increases in wealth.

[22]For an interesting comment on this paper, see Adda, Chandola and Marmot (2003).

While it may be tempting to say that our research is at loggerheads with this earlier work, we do not believe that this is the case. It is true that we do provide some evidence that income shocks may have sizable impacts on the health of working-aged men at the bottom of the income distribution. However, this is, by no means, in contradiction with the assertion that exogenous changes in wealth (not income) do not influence health in a population of older people.

It is also important to place this work within the context of the literature on recessions and health. First, we provide no solid evidence that health improves when economic circumstances deteriorate. Second, we provide ample evidence that poor aggregate and individual economic conditions are correlated with poor health. Third, we provide evidence that part of this correlation is causal.

Finally, some caveats on the limitations of this work deserve to be mentioned. First, it is not clear to what extent our estimates of the impact of labor income on self-reported health status translate into an impact on mortality. Given the results of Section 4, we believe that there may be some effect on mortality, but the magnitude of this effect is hard to infer from this analysis. For this reason, these results should not be used to conclude that Brenner's results were correct. Second, due to the constraints of the PSID, the health measures that we employ are somewhat limited. However, one of the primary advantages of the SRHS measures that we employ is that they exhibit significant variation across time which enables the use of panel data methods such as the AB estimator. Without substantial time variation, as would be the case with measures of specific conditions such as diabetes and heart disease, these methods cannot be used.

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Table 1: Summary Statistics

	Definition	Mean (Standard Deviation)	
		Men	Women
Age	Individual's Age	46.61 (14.09)	48.29 (15.04)
Black	= 1 if Black	0.26 (0.44)	0.30 (0.46)
No College	= 1 if Individual Never Went to College	0.61 (0.49)	0.68 (0.47)
SRHS	Self-Rated Health Status	2.49 (1.16)	2.71 (1.18)
Good Health	= 1 if SRHS <= 2	0.53 (0.50)	0.44 (0.50)
Bad Health	= 1 if SRHS >= 4	0.19 (0.40)	0.25 (0.43)
Unemployment Rate	County Level Unemployment Rate	6.33 (2.52)	6.40 (2.51)
Labor Income[1,2]	Individual's Labor Income	18277.45 (19513.65)	6746.01 (8808.71)
Zero Labor Supply[1]	= 1 if Labor Income = 0	0.16 (0.37)	0.38 (0.48)

*All summary statistics correspond to the years 1984 - 1993 except for labor income and supply.
**Summary statistics are for people older than 30.
[1]These summary statistics correspond to 1978 - 1993.
[2]Labor Income is in 1982 dollars.

Table 2: Mortality in the PSID

	% of Sample that Died Before 2003[1]	Average Survival in Years[2]
1984	22.83	17.66
1985	20.80	16.94
1986	19.22	16.19
1987	17.61	15.45
1988	15.82	14.71
1989	14.27	13.91
1990	11.25	13.15
1991	10.24	12.28
1992	8.89	11.42
1993	7.70	10.55

*All statistics correspond to people older than 30.

[1]Each number is the percentage of people who are at least 30 in a given year who die before 2003.

[2]Average survival is the average number of years subesquent to the survey year that the individual has survived.

Table 3: SRHS and Mortality in the PSID

	Between 30 and 60	Older Than 60
	Men	
Age	1.075	1.079
	(10.32)	(6.48)
Good Health	0.637	0.607
	(−3.17)	(−4.10)
Bad Health	2.114	1.167
	(5.93)	(1.27)
Likelihood	-2170.32	-2670.48
N	2522	667
	Women	
Age	1.087	1.084
	(10.85)	(10.70)
Good Health	0.747	0.922
	(−1.64)	(−0.67)
Bad Health	1.944	1.617
	(4.73)	(4.82)
Likelihood	-1896.37	-3623.49
N	2875	981

*This table contains results from the Cox-Proportional Hazard model.

**Each cell reports the hazard ratio for an incremental change in a given variable.

***t-ratios correspond to the unreported coefficients for each variable.

****All standard errors account for using county-level variables.

Table 4: Hazard Models- 1984 Base Year - Between 30 and 60

	(1)	(2)	(3)	(4)	(5)
			Men		
Age	1.090	1.087	1.076	1.102	1.056
	(13.55)	(12.82)	(10.70)	(1.06)	(0.59)
Age^2	-	-	-	1.000	1.000
				(−0.26)	(0.24)
Black	1.759	1.575	1.337	1.337	1.281
	(4.58)	(3.52)	(2.17)	(2.18)	(1.80)
No College	-	1.558	1.333	1.333	1.430
		(2.89)	(1.86)	(1.86)	(2.20)
Good Health	-	-	0.722	0.722	0.724
			(−2.23)	(−2.23)	(−2.17)
Bad Health	-	-	1.925	1.926	1.895
			(5.05)	(5.05)	(4.94)
Unemployment Rate	1.057	1.051	1.042	1.042	1.037
	(4.93)	(4.36)	(3.54)	(3.53)	(1.98)
State Dummies	No	No	No	No	Yes
Likelihood	-2186.58	-2172.70	-2152.33	-2152.28	-2119.74
N	2522	2518	2518	2518	2518
			Women		
Age	1.102	1.101	1.090	1.188	1.165
	(12.86)	(12.31)	(11.09)	(1.88)	(1.63)
Age^2	-	-	-	0.999	0.999
				(−0.94)	(−0.68)
Black	1.842	1.795	1.492	1.487	1.723
	(4.75)	(4.56)	(2.71)	(2.69)	(3.54)
No College	-	1.204	1.023	1.015	1.009
		(1.19)	(0.14)	(0.09)	(0.05)
Good Health	-	-	0.809	0.809	0.796
			(−1.12)	(−1.13)	(−1.23)
Bad Health	-	-	1.803	1.808	1.949
			(4.09)	(4.12)	(4.52)
Unemployment Rate	0.992	0.991	0.985	0.986	0.964
	(−0.39)	(−0.45)	(−0.74)	(−0.73)	(−1.44)
State Dummies	No	No	No	No	Yes
Likelihood	-1904.81	-1904.08	-1891.65	-1891.20	-1870.55
N	2875	2871	2871	2871	2871

*This table contains results from the Cox-Proportional Hazard model.

**Each cell reports the hazard ratio for an incremental change in a given variable.

***t-ratios correspond to the unreported coefficients for each variable.

****All standard errors account for using county-level variables.

Table 5: Probits- 1984 Base Year - Between 30 and 60

	(1)	(2)	(3)	(4)	(5)
			Men		
Age	0.008	0.008	0.007	0.002	−0.001
	(14.05)	(13.73)	(11.79)	(0.24)	(−0.13)
Age^2	-	-	-	0.000	0.000
				(0.58)	(0.97)
Black	0.069	0.054	0.037	0.037	0.029
	(4.61)	(3.59)	(2.47)	(2.48)	(1.88)
No College	-	0.042	0.028	0.028	0.034
		(3.20)	(2.15)	(2.12)	(2.47)
Good Health	-	-	−0.032	−0.032	−0.031
			(−2.34)	(−2.34)	(−2.21)
Bad Health	-	-	0.082	0.082	0.080
			(4.89)	(4.87)	(4.78)
Unemployment Rate	0.006	0.005	0.004	0.004	0.004
	(4.65)	(4.11)	(3.43)	(3.43)	(1.88)
State Dummies	No	No	No	No	Yes
Pseudo - R^2	0.1117	0.1186	0.1394	0.1397	0.1591
N	2532	2528	2528	2528	2425
			Women		
Age	0.007	0.007	0.006	0.006	0.005
	(13.75)	(13.15)	(11.79)	(0.95)	(0.79)
Age^2	-	-	-	0.000	0.000
				(0.10)	(0.22)
Black	0.052	0.050	0.034	0.034	0.041
	(4.95)	(4.79)	(3.08)	(3.08)	(3.49)
No College	-	0.012	0.002	0.002	0.000
		(1.13)	(0.14)	(0.14)	(0.01)
Good Health	-	-	−0.014	−0.014	−0.013
			(−1.13)	(−1.13)	(−1.15)
Bad Health	-	-	0.053	0.054	0.058
			(4.43)	(4.43)	(4.91)
Unemployment Rate	−0.001	−0.001	−0.001	−0.001	−0.002
	(−0.67)	(−0.77)	(−1.03)	(−1.03)	(−1.25)
State Dummies	No	No	No	No	Yes
Pseudo - R^2	0.1315	0.1319	0.1468	0.1468	0.1679
N	2890	2886	2886	2886	2856

*This table contains results from probits where the dependent variable is an indicator for dying between 1984 and 2003.

**Each cell reports marginal effects.

***t-ratios correspond to the unreported coefficients for each variable.

****All standard errors account for using county-level variables.

Table 6: Hazard Models- 1984 Base Year - 60 Years or Older

	(1)	(2)	(3)	(4)	(5)
			Men		
Age	1.081	1.081	1.080	1.388	1.445
	(6.78)	(6.74)	(6.42)	(1.95)	(1.93)
Age^2	-	-	-	0.998	0.998
				(−1.43)	(−1.45)
Black	1.201	1.168	1.066	1.137	1.280
	(1.71)	(1.47)	(0.56)	(1.08)	(1.53)
No College	-	1.166	0.989	0.991	0.963
		(1.21)	(−0.09)	(−0.07)	(−0.27)
Good Health	-	-	0.605	0.601	0.613
			(−4.06)	(−3.96)	(−3.46)
Bad Health	-	-	1.140	1.158	1.201
			(1.10)	(1.26)	(1.43)
Unemployment Rate	1.029	1.026	1.026	1.023	1.019
	(3.13)	(2.72)	(2.71)	(2.18)	(1.10)
State Dummies	No	No	No	No	Yes
Likelihood	-2683.88	-2683.13	-2668.75	-2664.63	-2650.08
N	667	667	667	667	667
			Women		
Age	1.085	1.085	1.085	1.259	1.203
	(8.87)	(8.69)	(9.97)	(2.01)	(1.86)
Age^2	-	-	-	0.999	0.999
				(−1.27)	(−0.98)
Black	1.159	1.120	0.997	1.037	1.127
	(1.25)	(0.95)	(−0.02)	(0.33)	(1.09)
No College	-	1.347	1.198	1.204	1.175
		(2.42)	(1.45)	(1.49)	(1.20)
Good Health	-	-	0.946	0.936	0.963
			(−0.46)	(−0.57)	(−0.32)
Bad Health	-	-	1.600	1.549	1.630
			(4.33)	(4.58)	(5.38)
Unemployment Rate	1.009	1.007	1.003	1.003	1.044
	(0.75)	(0.59)	(0.26)	(0.22)	(1.93)
State Dummies	No	No	No	No	Yes
Likelihood	-3641.26	-3631.35	-3615.34	-3613.29	-3590.08
N	981	979	979	979	979

*This table contains results from the Cox-Proportional Hazard model.

**Each cell reports the hazard ratio for an incremental change in a given variable.

***t-ratios correspond to the unreported coefficients for each variable.

****All standard errors account for using county-level variables.

Table 7: Hazard Models- 1984 Base Year - Between 30 and 60 - Restricted Sample

	(1)	(2)	(3)	(4)	(5)
			Men		
Age	1.083	1.078	1.067	1.075	1.036
	(12.38)	(11.25)	(9.25)	(0.85)	(0.41)
Age^2	-	-	-	1.000	1.000
				(−0.09)	(0.39)
Black	2.459	2.059	1.652	1.653	1.328
	(8.52)	(6.11)	(3.82)	(3.85)	(2.11)
No College	-	1.864	1.585	1.585	1.738
		(4.01)	(2.89)	(2.89)	(3.40)
Good Health	-	-	0.678	0.678	0.693
			(−2.77)	(−2.76)	(−2.50)
Bad Health	-	-	2.020	2.018	2.057
			(5.30)	(5.33)	(5.20)
Unemployment Rate	1.061	1.051	1.041	1.041	1.038
	(4.55)	(3.61)	(3.40)	(3.40)	(1.79)
State Dummies	No	No	No	No	Yes
Likelihood	-2022.61	-2004.19	-1979.72	-1979.71	-1945.70
N	1507	1503	1503	1503	1503
			Women		
Age	1.095	1.092	1.080	1.199	1.170
	(12.65)	(11.86)	(10.29)	(1.91)	(1.60)
Age^2	-	-	-	0.999	0.999
				(−1.10)	(0.76)
Black	2.436	2.287	1.790	1.786	2.034
	(6.83)	(6.31)	(3.80)	(3.81)	(4.64)
No College	-	1.455	1.223	1.212	1.186
		(2.38)	(1.23)	(1.19)	(1.04)
Good Health	-	-	0.752	0.748	0.753
			(−1.58)	(−1.61)	(−1.53)
Bad Health	-	-	1.825	1.815	1.922
			(4.41)	(4.39)	(4.57)
Unemployment Rate	0.985	0.982	0.977	0.977	0.955
	(−0.77)	(−0.92)	(−1.18)	(−1.18)	(−1.93)
State Dummies	No	No	No	No	Yes
Likelihood	-1763.83	-1761.09	-1747.23	-1746.57	-1722.96
N	1723	1719	1719	1719	1719

*This table contains results from the Cox-Proportional Hazard model.

**Each cell reports the hazard ratio for an incremental change in a given variable.

***t-ratios correspond to the unreported coefficients for each variable.

****All standard errors account for using county-level variables.

Table 8: Selection Probits - Between 30 and 60

	(1)	(2)	(3)	(4)	(5)
			Men		
Age	−0.015	−0.018	−0.018	0.035	0.043
	(−4.85)	(−5.45)	(−5.38)	(0.94)	(1.17)
Age^2	-	-	-	−0.001	−0.001
				(−1.42)	(−1.65)
Black	0.338	0.260	0.251	0.253	0.144
	(4.79)	(3.47)	(3.35)	(3.37)	(1.52)
No College	-	0.292	0.281	0.286	0.273
		(5.00)	(4.96)	(4.94)	(4.78)
Good Health	-	-	−0.087	−0.087	−0.065
			(−1.52)	(−1.51)	(−1.05)
Bad Health	-	-	−0.059	−0.053	−0.060
			(−0.75)	(−0.66)	(−0.72)
Unemployment Rate	−0.003	−0.007	−0.007	−0.008	−0.008
	(−0.26)	(−0.73)	(−0.76)	(−0.77)	(−0.64)
State Dummies	No	No	No	No	Yes
PseudoR^2	0.0204	0.0287	0.0293	0.0300	0.0615
N	2532	2528	2528	2528	2516
			Women		
Age	−0.011	−0.014	−0.015	0.023	0.012
	(−4.79)	(−5.89)	(−5.96)	(0.73)	(0.38)
Age^2	-	-	-	−0.000	−0.000
				(−1.22)	(−0.87)
Black	0.382	0.338	0.318	0.319	0.261
	(5.43)	(4.64)	(4.29)	(4.29)	(3.15)
No College	-	0.287	0.263	0.261	0.274
		(5.48)	(4.62)	(4.57)	(4.59)
Good Health	-	-	−0.123	−0.234	−0.110
			(−1.77)	(−1.78)	(−1.60)
Bad Health	-	-	−0.038	−0.037	−0.064
			(−0.57)	(−0.54)	(−1.02)
Unemployment Rate	−0.004	−0.008	−0.009	−0.009	−0.011
	(−0.41)	(−0.87)	(−0.90)	(−0.90)	(−0.95)
State Dummies	No	No	No	No	Yes
PseudoR^2	0.0202	0.0271	0.0283	0.0287	0.0564
N	2890	2886	2886	2886	2880

*This table contains results from probits where the dependent variable is the selection indicator described in Section 4.2.

**t-ratios are reported below each coefficient.

***All standard errors account for using county-level variables.

Table 9: Random Effects Estimates - Male Mortality

	(1)	(2)	(3)
Between 30 and 60			
Death Occurred	1 Year After	3 Years After	5 Years After
Age	0.026	0.059	0.051
	(8.46)	(8.26)	(7.77)
Black	0.567	0.734	0.655
	(8.23)	(5.22)	(5.01)
No College	0.041	0.154	0.191
	(0.66)	(1.22)	(1.68)
Good Health	−0.08	−0.178	−0.272
	(−1.20)	(−1.63)	(−2.76)
Bad Health	0.567	0.525	0.237
	(8.23)	(4.71)	(2.26)
Unemployment Rate	0.038	−0.006	0.004
	(4.29)	(−0.37)	(0.23)
Likelihood	-1292.52	-1246.78	-1192.84
N	6315	6315	6315
Older Than 60			
Death Occurred	1 Year After	3 Years After	5 Years After
Age	0.117	0.067	0.036
	(12.32)	(12.95)	(9.82)
Black	0.533	0.253	0.158
	(3.88)	(3.08)	(2.43)
No College	−0.169	−0.071	−0.078
	(−1.25)	(−0.82)	(−1.16)
Good Health	−0.098	−0.300	−0.114
	(−0.91)	(−3.83)	(−1.74)
Bad Health	0.704	0.257	0.053
	(7.89)	(4.06)	(0.92)
Unemployment Rate	0.029	−0.018	−0.012
	(1.82)	(−1.57)	(−1.25)
Likelihood	-1831.30	-2225.95	-2202.63
N	1656	1656	1656

*This table contains results from random effects probits where the dependent variables are indicators for dying between the survey year and one, three and five years after.

**Each cell reports marginal effects.

***t-ratios correspond to the unreported coefficients for each variable.

Table 10: Random Effects Estimates - Female Mortality

	(1)	(2)	(3)
Between 30 and 60			
Death Occurred	1 Year After	3 Years After	5 Years After
Age	0.092	0.088	0.070
	(6.13)	(8.67)	(7.25)
Black	1.219	1.217	1.012
	(4.99)	(6.18)	(5.14)
No College	−0.150	0.014	0.081
	(−0.72)	(0.08)	(0.54)
Good Health	−0.232	−0.126	0.022
	(−1.22)	(−0.87)	(0.17)
Bad Health	0.881	0.576	0.367
	(5.24)	(4.27)	(2.96)
Unemployment Rate	−0.030	−0.008	−0.004
	(−1.04)	(−0.34)	(−0.18)
Likelihood	-728.63	-922.62	-961.10
N	6923	6923	6923
Older Than 60			
Death Occurred	1 Year After	3 Years After	5 Years After
Age	0.147	0.076	0.048
	(11.13)	(13.97)	(13.24)
Black	0.594	0.247	0.072
	(3.77)	(2.84)	(1.09)
No College	0.077	0.184	0.092
	(0.43)	(1.67)	(1.13)
Good Health	−0.173	−0.068	−0.166
	(−1.39)	(−0.80)	(−2.33)
Bad Health	0.644	0.294	0.109
	(6.33)	(4.19)	(1.87)
Unemployment Rate	−0.015	−0.012	−0.015
	(−0.81)	(−0.96)	(−1.41)
Likelihood	-1930.79	-2378.60	-2430.73
N	2194	2194	2194

*This table contains results from random effects probits where the dependent variables are indicators for dying between the survey year and one, three and five years after.

**Each cell reports marginal effects.

***t-ratios correspond to the unreported coefficients for each variable.

Table 11: Fixed Effects Estimates - SRHS

	(1)	(2)	(3)	(4)
	Men			
	Older Than 60		*Between 30 and 60*	
Dependent Variable	Good Health	Bad Health	Good Health	Bad Health
Age	−0.101	0.114	−0.094	0.105
	(−7.73)	(8.63)	(−14.44)	(10.57)
Unemployment Rate	0.022	0.015	−0.026	0.005
	(1.08)	(0.72)	(−2.45)	(0.31)
Likelihood	-1790.36	-1700.52	-7253.74	-3119.23
N^+	682	652	2826	1304
	Women			
	Older Than 60		*Between 30 and 60*	
Dependent Variable	Good Health	Bad Health	Good Health	Bad Health
Age	−0.047	0.050	−0.052	0.051
	(−4.13)	(4.48)	(−8.49)	(5.92)
Unemployment Rate	−0.006	0.000	−0.007	0.017
	(−0.30)	(0.01)	(−0.75)	(1.31)
Likelihood	-2232.85	-2286.71	-8094.96	-4168.36
N^+	835	851	3076	1714

*This table contains results from fixed-effects probits where the dependent variable is an indicator for good or bad health.
**t-ratios correspond to the unreported coefficients for each variable.
$^+$N corresponds to the "effective" sample size i.e. the total number of people who change health states while in the sample.

Table 12: Macroeconomic Shocks and Labor Market Outcomes

	Labor Income	Labor Supply
1% Increase in Unemployment	−0.030	0.001
Men	(−5.05)	(1.39)
1% Increase in Unemployment	−0.028	0.000
Women	(−3.55)	(0.32)

*This table reports the coefficient on unemployment from fixed-effects regressions where the dependent variables are labor income and labor supply. All regressions contain a polynomial in age. The regressions where estimated using people between the ages of 30 and 60.
**t-statistics in parentheses.
***Each cell reports the effects of a 1% increase in unemployment on labor income and labor force participation.

Table 13: Arellano-Bond Estimates - Men Between Ages 30 and 60

	(1)	(2)	(3)	(4)
	Predetermined Variables			
Dependent Var	Bad	Bad	Bad	SRHS
Lagged Health	0.010	0.093	0.095	0.103
	(9.55)	(9.06)	(9.31)	(9.66)
Age	0.005	0.005	0.005	0.017
	(2.79)	(2.82)	(2.93)	(3.43)
Zero Labor Income?[1]	-	0.037	−0.039	0.170
		(2.31)	(−0.81)	(3.57)
Labor Income	−0.006	-	−0.010	-
	(−2.94)		(−1.65)	
m_1^2	−61.23	−60.64	−61.62	−59.25
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	−0.46	−0.76	−0.65	1.64
	(0.645)	(0.446)	(0.517)	(0.102)
Two-Step Sargan[2]	165.34	143.74	252.22	192.30
	(0.011)	(0.133)	(0.051)	(0.000)
O.I. Restrictions	126	126	217	126
	Endogenous Variables			
Lagged Health	0.100	0.095	0.096	0.107
	(9.54)	(9.08)	(9.32)	(9.86)
Age	0.005	0.005	0.005	0.015
	(2.77)	(2.64)	(2.77)	(2.98)
Zero Labor Income?[1]	-	0.059	−0.051	0.453
		(1.35)	(−0.32)	(3.39)
Labor Income	−0.006	-	−0.011	-
	(−0.97)		(−0.65)	
m_1^2	−58.65	−58.30	−60.22	−55.06
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	−0.43	−0.64	−0.60	1.89
	(0.666)	(0.523)	(0.551)	(0.058)
Two-Step Sargan[2]	149.27	120.06	230.20	181.30
	(0.027)	(0.430)	(0.077)	(0.000)
O.I. Restrictions	118	118	201	118
N	5336	5336	5336	5336

*t-statistics reported below each coefficient estimate.

[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.

[2]p-values in parentheses.

Table 14: Arellano-Bond Estimates - Women Between Ages 30 and 60

	(1)	(2)	(3)	(4)
	Predetermined Variables			
Dependent Var	Bad	Bad	Bad	SRHS
Lagged Health	0.087	0.089	0.088	0.060
	(9.37)	(9.56)	(9.56)	(6.39)
Age	0.012	0.012	0.012	0.029
	(6.12)	(6.12)	(6.12)	(5.61)
Zero Labor Income?[1]	-	0.001	−0.005	0.042
		(0.13)	(−0.14)	(1.56)
Labor Income	0.000	-	−0.001	-
	(0.01)		(−0.19)	
m_1^2	−67.23	−67.61	−67.92	−63.15
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	1.49	1.56	1.53	2.49
	(0.135)	(0.119)	(0.125)	(0.0123)
Two-Step Sargan[2]	141.20	123.72	222.87	199.31
	(0.168)	(0.541)	(0.378)	(0.000)
O.I. Restrictions	126	126	217	126
	Endogenous Variables			
Lagged Health	0.087	0.089	0.088	0.059
	(9.23)	(9.46)	(9.48)	(6.29)
Age	0.012	0.012	0.012	0.029
	(6.17)	(6.07)	(6.08)	(5.67)
Zero Labor Income?[1]	-	−0.001	0.311	−0.010
		(−0.03)	(2.38)	(−0.15)
Labor Income	0.002	-	0.036	-
	(0.73)		(2.47)	
m_1^2	−66.34	−66.72	−64.78	−62.52
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	1.44	1.55	1.80	2.41
	(0.151)	(0.122)	(0.072)	(0.016)
Two-Step Sargan[2]	139.40	119.16	205.39	192.97
	(0.087)	(0.453)	(0.401)	(0.000)
O.I. Restrictions	118	118	201	118
N	5776	5776	5776	5776

*t-statistics reported below each coefficient estimate.

[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.

[2]p-values in parentheses.

Table 15: Arellano-Bond Estimates - Income by Quartile, People Between 30 and 60

	(1)	(2)	(3)	(4)	(5)
	Men			Women	
Lagged Health	0.093	0.103	0.101	0.059	0.090
	(9.06)	(10.06)	(9.77)	(9.56)	(9.62)
Age	0.005	0.005	0.005	0.012	0.012
	(2.82)	(2.95)	(2.93)	(6.12)	(6.11)
Zero Labor Income?[1]	0.037	-	-	0.001	-
	(2.31)			(0.13)	
Income 25 Percentile	-	0.004	-	-	-
		(0.38)			
Income 75 Percentile	-	-	0.001	-	0.004
			(0.05)		(0.32)
m_1^2	−60.64	−62.02	−61.65	−67.61	−67.99
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	−0.76	−0.41	−0.50	1.56	1.58
	(0.446)	(0.679)	(0.615)	(0.119)	(0.114)
Two-Step Sargan[2]	143.74	145.01	154.65	123.72	134.79
	(0.133)	(0.118)	(0.042)	(0.541)	(0.280)
O.I. Restrictions	126	126	126	126	126
N	5336	5336	5336	5776	5776

*This table assumes that all income and labor supply variables are predetermined.
**t-statistics reported below each coefficient estimate.
[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.
[2]p-values in parentheses.

Figure 1: Survivor Functions in the PSID